

Attorney Docket No.: PTQ-0028
Inventors: Van Eyk et al.
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REMARKS

Claims 1-7, 16-18, 20-27, 31, 34, 35 and 37-41 are pending in the instant application. Claims 1-7, 16-18, 20-27, 31, 34, 35 and 37-41 have been rejected.

Reconsideration is respectfully requested in light of the following remarks.

Rejection of Claims under 35 U.S.C. 103(a)

Claims 1, 16-18, 20-27, 31 and 34 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Takahashi et al. (Clinical Biochemistry, Vol. 29, No. 4, August 1996, pages 301-308) in view of Solaro et al. (Journal of Molecular Cell Cardiology, Vol. 28, pages 217-230, 1996) and Lin et al. (The Journal of Biological Chemistry, Vol. 271, No. 1, 1/5/1996, pages 244-249) and further in view of Han et al. (International Journal of Biochemistry, Vol. 24, No. 1, 1992, pages 19-28).

Claims 2-7, 28, 34-35, 38 and 40-41 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Takahashi et al. (Clinical Biochemistry, Vol. 29, No. 4, August 1996, pages 301-308) in view of Solaro et al. and Lin et al. and further in view of Han et al. as applied to claims 1, 16-18, 20-27 and 34 above, and further in view of Wicks et al. (U.S. Patent 5,834,220).

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It is respectfully pointed out that claim 28 is no longer pending.

Claims 37 and 39 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Takahashi et al. (Clinical Biochemistry, Vol. 29, No. 4, August 1996, pages 301-308) in view of Solaro et al. and Lin et al. and further in view of Han et al. and Wicks et al. (U.S. Patent 5,834,220) as applied to claims 2-7, 28, 34-35, 38 and 40-41 above, and further in view of Jideama et al. (The Journal of Biological Chemistry, Vo. 271, No. 38, 9/20/96, pages 23277-23283).

Applicants respectfully traverse these rejections.

As acknowledged by the Examiner in the Office Action mailed November 30, 2007, Solaro et al. taught changes in **cardiac** function, Lin et al. measured covalent binding of peptides to **cardiac** troponin C and Wicks et al. taught assaying for **cardiac** troponin I along with **cardiac** troponin C. Teachings of Jideama also relate to **cardiac** troponin I and **cardiac** troponin T in the myocardium. See Abstract. Clearly, Solaro et al., Lin et al., Wicks et al. and Jideama relate to **cardiac** myofilament proteins, **not skeletal** myofilament proteins. Han et al. is cited for its general teachings of post-translational modification being recognized in a wide variety of cell types. Thus,

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Applicants respectfully disagree with the Examiner that the skilled artisan would combine teachings of cardiac specific references with Takahashi et al., which teaches measuring skeletal troponin I (fsTnI) in skeletal muscle damage.

Further, the suggested combination of references provides no reasonable expectation of success with respect to the instant claimed invention that evaluating for the presence of a chemical adduct of a myofilament protein selected from the group consisting of skeletal troponin I, skeletal troponin T and myosin light chain 1 would be indicative of skeletal muscle damage.

The Examiner has acknowledged that Takahashi et al. differs from the instant invention in not specifically teaching myofilament protein modification products. The Examiner has further acknowledged that Takahashi et al. differs from the instant invention in not specifically detecting a chemical adduct of the myofilament protein modification product. The Examiner has also acknowledged that Takahashi et al. is silent with respect to the myofilament protein modification product being a post-translational modification.

The secondary references of Solaro et al., Lin et al., Wicks et al. and Jideama cannot remedy the deficiencies of Takahashi et al. as these reference relate to **cardiac**

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myofilament proteins and **cardiac** function only, not **skeletal** myofilament proteins nor **skeletal** muscle damage as claimed. Han also fails to remedy deficiencies in the primary reference of Takahashi et al. as this general teaching is not specific to skeletal myofilament proteins or skeletal muscle damage.

Applicants believe the failure of these cardiac related references to remedy deficiencies in the teachings of Takahashi et al. is evidenced by the Examiner's withdrawal in the instant Office Action of the prior obviousness rejections raised in the Office Action dated November 30, 2007 upon amendment of the claims to skeletal muscle damage.

MPEP 2143.02 and the case law are clear; a rationale to support a conclusion that a claim would have been obvious is that all the claimed elements were known in the prior art and one skilled in the art could have combined the elements as claimed by known methods with no change in their respective functions, and the combination would have yielded nothing more than predictable results to one of ordinary skill in the art. *KSR International Co. v. Teleflex Inc.*, 550 U.S. ___, ___, 82 USPQ2d 1385, 1395 (2007); *Sakraida v. AG Pro, Inc.*, 425 U.S. 273, 282, 189 USPQ 449, 453 (1976); *Anderson's-Black Rock, Inc. v. Pavement Salvage Co.*, 396

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U.S. 57, 62-63, 163 USPQ 673, 675 (1969); *Great Atlantic & P. Tea Co. v. Supermarket Equipment Corp.*, 340 U.S. 147, 152, 87 USPQ 303, 306 (1950). In the instant case, however, the claimed elements of myofilament protein modification products being a chemical adduct of a myofilament protein selected from the group consisting of skeletal troponin I, skeletal troponin T and myosin light chain 1 and their association with skeletal muscle damage were **not** known in the prior art. One skilled in the art could **not** have combined the methods of Solaro et al., Lin et al., Wicks et al., Jideama and Han relating to cardiac myofilament proteins and cardiac function, with no change in their respective functions, with Takahashi et al., acknowledged by the Examiner to not teach myofilament protein modification products, detecting a chemical adduct of the myofilament protein modification product, or myofilament protein modification product being a post-translational modification, to arrive at the instant invention. Results of the instant invention were clearly **not** predictable to one of ordinary skill in the art from the cited combined teachings as none of these references teach or suggest the existence of myofilament protein modification products being a chemical adduct of a myofilament protein selected from the

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group consisting of skeletal troponin I, skeletal troponin T and myosin light chain 1 or their association with skeletal muscle damage.

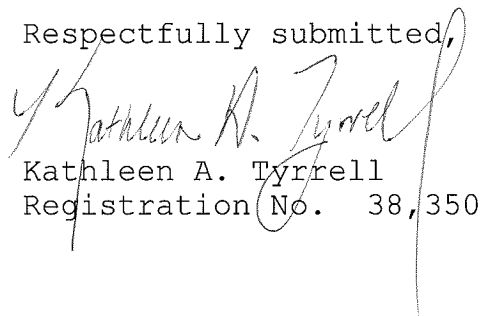
Thus, a proper rationale to support a conclusion that the instant claimed invention would have been obvious is **not** provided by the combinations of cited references. See MPEP 2143.02.

Withdrawal of these rejections under 35 U.S.C. 103(a) is therefore respectfully requested.

Conclusion

Applicants believe that the foregoing comprises a full and complete response to the Office Action of record. Accordingly, favorable reconsideration and subsequent allowance of the pending claims is earnestly solicited.

Respectfully submitted,


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